

Annotated Bibliography

Which exercise modes and intensities contribute effectively to the treatment of Dyslipidemia?

A comprehensive review of this question was undertaken by Durstine et al(2002). Subsequent original research papers selected for this bibliography supplement and refine the understandings summarised in Durstine et al (2002). The selected papers focus on the special requirements of population sub groups and alternate exercise modalities.

Angelopoulos T, Sivo S, Kyriazis G, Caplan J, Zoeller R, Lowndes J, et al. Do age and baseline LDL cholesterol levels determine the effect of regular exercise on plasma lipoprotein cholesterol and apolipoprotein B levels? European Journal of Applied Physiology. 2007;101(5):621-8. DOI: 10.1007/s00421-007-0537-1

This study was essentially a longitudinal study of 41 participants, ranging in age from 18 to 59 years, who all agreed to undertake 6 months of supervised aerobic exercise training, 4 times per week at 60 to 85% of maximal heart rate. Participants used treadmills and stationary cycles. The training sessions increased from 15 to 40 minutes over the first 4 weeks and remained at 40 minutes for the remainder of the study period.

Apolipoprotein B (ApoB) is a constituent of VLDLs and LDLs and is now widely used as a predictor of coronary heart disease risk as it indicates the presence of the VLDL and the LDL sub-class that are linked to atherogenic processes. The authors note a high unexplained variability in ApoB responses among previous aerobic exercise training studies. In this study, they sought to determine whether age or baseline lipid levels influenced the ApoB response to aerobic exercise.

Over the entire study cohort they found neither ApoB response to aerobic exercise, nor any significant effect of aerobic exercise training on any anthropometric measure, lipid or lipoprotein. However, they did identify some interesting differences in response between sub-groups with low and high baseline LDL-C levels.

- low LDL-C ≤ 3.4 mmol/L - 19 participants
- high LDL-C > 3.4 mmol/L - 22 participants

The threshold selected equates to the NCEP LDL-C threshold of 130 mg/dL. Both groups displayed a significant positive effect of aerobic exercise on body weight and $\text{VO}_{2\text{max}}$ (both absolute and relative). But for lipid profiles, the chronic effects of exercise were opposite. The low LDL-C group had a significant increase in LDL-C and TC whereas the high LDL-C group had a significant decrease in LDL-C and TC. For the high LDL-C group, mean LDL-C concentration was 4.3 mmol/L pre-training, 3.9 mmol/L at 24hrs post training and 4.1 mmol/L 72hrs after the last session. This result suggests that for those with elevated LDL-C, frequent aerobic exercise training may be most beneficial to maximise the acute effects in lowering LDL-C levels.

Analysis of variance revealed that age had no effect on ApoB nor lipoprotein response to exercise.

There are two methodological issues with this study –

- it provides very poor quantification of the exercise load applied. The intensity was between 60 and 85% of HR_{max} - for the average subject, at 40 years of age, this equates anywhere between 115bpm and 155bpm.
- dietary intake was quantified by telephone interview and 24 hour dietary recall on two occasions prior to commencement and on two occasions during the last month of the study. The authors

note no significant change in dietary macronutrient composition but fail to report any temporal change in the types of dietary fats consumed.

The other unusual finding here is that 6 months of aerobic training resulted in a significant increase in LDL-C, and TC, in those subjects with “normal” LDL-C levels.

Bey L, Hamilton MT. Suppression of skeletal muscle lipoprotein lipase activity during physical inactivity: a molecular reason to maintain daily low-intensity activity. The Journal of Physiology. 2003;551(2):673. DOI: 10.1113/jphysiol.2003.045591

This paper takes a more mechanistic approach to exercise effect on lipid profiles than the other papers considered in this bibliography. The target of the study is lipoprotein lipase (LPL), an enzyme critical to the tissue-specific uptake of triglyceride rich lipoproteins by non-hepatic tissue.

This randomised controlled trial investigates the effect of inactivity on the LPL levels of the soleus and quadriceps muscles of rats (and of mice). In the “chronic” study, the researchers immobilised the hind legs of rats for 10 hours per day for up to 11 days, using a technique referred to as hind-leg unloading (HU). This treatment resulted in no atrophy of the soleus muscle over the study period.

Tissue samples of soleus and quadriceps muscles were analysed from both control and HU rats and significant reductions in LPL activity were observed in the HU rats after 11 days. HDL-C was also significantly reduced after 1 and 11 days of HU treatment. The study was repeated with mice to the same effect. They also found no reduction in LPL in the heart or other muscle of rats treated with hind-leg unloading.

In the “acute” study, they again immobilised the hind-legs of a sample of rats for 12 hours, followed by a 4 hour “reloading” period when the rats were returned to normal quadrupedal locomotion involving low speed treadmill walking. They found that LPL activity was raised 8-fold with the low intensity activity and returned to control levels in 4 hours.

Much of the discussion focuses on the genetic mechanisms that underpin the observed effect. But more importantly, from the point of view of this bibliography, the authors make the argument that their study lends support to the epidemiological studies that underpin the ACSM exercise guidelines. They particularly note that their research lends support to the notion that the “greatest benefit would come from getting the least active to become moderately active”.

Durstine JL, Grandjean PW, Davis PG, Ferguson MA, Alderson NL, DuBose KD. Blood lipid and lipoprotein adaptations to exercise: a quantitative analysis. Sports Medicine. 2001;31(15):1033-62.

These researchers are prolific contributors to original research and reviews in the field of exercise and lipid metabolism.

This 2001 review includes reference to 157 papers – both cross-sectional studies and exercise interventions.

The cross sectional studies suggest that “most sedentary individuals will experience elevations of 3.5 to 6 mg/dl in HDL-C by increasing their exercise energy expenditure to 1500 to 2200 kcal/wk.” These studies also suggest that energy expenditure of 1500 to 2200 kcal/wk is associated with a reduction in Tg of between 7 to 20 mg/dl with further reductions as training volume is increased. There is little support in the cross-sectional literature for significant differences in TC and LDL-C between active and inactive groups, independent of bodyweight and body fat differences.

Those exercise interventions reviewed tended to confirm the observations from the cross sectional studies, that -

- aerobic exercise training infrequently affects TC and LDL-C in either men or women, but that in combination with a reduction in dietary fat intake, and/or weight loss, exercise training programmes have been observed to affect these cholesterol levels;
- HDL-C and Tg levels are more responsive to regular exercise than TC and LDL-C;
- a dose-response relationship between aerobic exercise and Tg and HDL-C levels is influenced most by training volume;
- resistance training does not seem to alter blood lipid and lipoprotein levels.

The authors make an interesting observation about acute versus chronic response to exercise interventions. They caution that when interpreting observed changes in lipid levels, particularly with respect to Tg levels, that the timing of a final blood sample after the final exercise session can be critical. They note that "reported changes after exercise training maybe a function of when the final blood sample was obtained relative to the last exercise session, rather than a cumulative effect of the training regimen."

WARNING - Most studies referred to in this bibliography, and more generally in the literature, use the Friedewald formula. Blood samples are commonly analysed for [TC], [HDL-C] and [Tg]. And then researchers have traditionally used Friedewald formula to estimate [VLDL-C] (viz. $[VLDL-C] = [Tg] \div 5$). LDL-C is then estimated as $[LDL-C] = [TC] - [HDL-C] - \text{estimated } [VLDL-C]$. Hence a biased estimation of exercise effect on Tg level can also compromise findings with respect to LDL-C.

Elliott K, Sale C, Cable N. Effects of resistance training and detraining on muscle strength and blood lipid profiles in postmenopausal women. British Journal of Sports Medicine. 2002;36(5):340.

After menopause, changes in hormonal levels are associated with adverse effects on serum lipids and lipoproteins (Sacks and Walsh 1990). The concentration of LDL-C increases and the concentration of 'protective' HDL-C decreases; and the rate of increase of the incidence of CHD rises exponentially in women after 55 years of age.

This randomised controlled trial, involving only 15 subjects, sought to identify the effects of resistance exercise training on muscle strength and blood lipid profiles of healthy, sedentary, postmenopausal women. The subjects, ranging in age from 49 to 62 years, were randomly assigned to a training group (TG) and a control group (CG), matched for years post menopause.

All subjects were assessed for muscle strength and blood lipid concentrations at

- baseline,
- after the 8 week period during which the TG undertook a resistance exercise program, and
- after a further 8 weeks to enable assessment of a detraining effect on the TG.

Strength assessment and resistance training were undertaken on weight machines under supervision. 10 repetition maximum (10RM) strength was assessed for bilateral knee flexion and extension, leg press, bench press, and lat pulldown,

During the 8-week intervention period, the TG undertook 3 resistance exercise training sessions per week. Each session involved a self-paced warmup on a cycle ergometer, followed by a 10 repetition warm-up at 50% of 10RM. Then each subject performed 3 sets of 8 repetitions of each exercise at 80% of their 10RM, with a two minute rest between sets.

Blood samples were analysed for TC, Tg and HDL-C. Friedewald's equation were used to calculate LDL-C and VLDL-C.

After 8 weeks of resistance exercise training, muscle strength increased significantly in the TG compared with baseline and the control group but produced no significant effect on blood lipid profiles. Muscle

strength remained significantly elevated above baseline after the 8 week detraining period but was trending back toward baseline values.

Blood lipid concentrations were unaffected by 8 weeks of resistance training. Body composition and blood pressure were similarly unaffected.

The authors noted that previous studies involving longer and more intense resistance exercise interventions, or aerobic exercise interventions achieving weight loss, had produced significantly positive effect on lipid profiles. They also note the downward trend in blood pressure (albeit not a significant trend).

The statistical power of this study was clearly constrained by the very small sample size. The authors commented on the relatively high risk of a Type II error in interpretation of the results and suggested that longer training programmes at higher work intensities may be needed to evoke significant changes in blood lipid profiles, body composition and blood pressure.

The authors contrast their findings with those of Prabhakaran et al (1999) in young women. They suggest that the findings of effect on TC and LDL-C in the earlier study were due to a higher intensity and longer duration of the exercise intervention. While acknowledging that the age difference between the sample groups may have affected exercise intensity, the authors ignore the likely effect of the different hormonal status of the study groups and consequent effect on lipid metabolism.

In most studies of the effect of exercise on lipid profiles, the researchers seek to minimise the confounding effect of changes in dietary intake during the study period. These authors have paid no regard to the effect of diet at all.

References

Prabhakaran B, Dowling EA, Branch JD, Swain DP, Leutholtz BC. Effect of 14 weeks of resistance training on lipid profile and body fat percentage in premenopausal women. *British Journal of Sports Medicine*. 1999;33(3):190.

Sacks FM, Walsh BW. The Effects of Reproductive Hormones on Serum Lipoproteins: Unresolved Issues in Biology and Clinical Practice. *Annals of the New York Academy of Sciences*. 1990;592(1):272-85. DOI: 10.1111/j.1749-6632.1990.tb30339.x

LeMura LM, von Duvillard SP, Andreacci J, Klebez JM, Chelland SA, Russo J. Lipid and lipoprotein profiles, cardiovascular fitness, body composition, and diet during and after resistance, aerobic and combination training in young women. *European Journal of Applied Physiology*. 2000;82(5):451-8. DOI: 10.1007/s004210000234

This randomised controlled trial investigated the effect of three exercise prescriptions on cardiovascular fitness, blood lipid profiles and body composition on young, healthy, sedentary women over a 16 week intervention period. 6 weeks after completion of the exercise intervention, all subjects were re-assessed.

The researchers recruited 48 healthy young women who were non-smokers and none were using any medications known to alter lipid metabolism, including oral contraceptives.

Each participant undertook preliminary assessment for anthropometric measures, blood lipid profile and cardiovascular fitness by maximal exercise test on a motor driven treadmill to determine baseline $\dot{V}O_{2max}$. This assessment was repeated after 8 weeks, 16 weeks (at completion of the exercise intervention) and finally at 6 weeks after completion of the intervention.

The participants were randomly allocated to one of four groups –

- aerobic training group (ATG) –training 3 times per week on non-consecutive days, initially for 30 minutes of continual exercise at 70 to 75% of HRmax. Mode was self-selected from cycle

ergometer, rower or treadmill. At week 8, after re-assessment, the exercise prescription was adjusted. Adjustments included intensity (up to 85% of HR_{max}), duration (up to 45 mins) and frequency (up to 4 /week)

- resistance training group (RTG) –training 3 times per week on non-consecutive days, initially doing 2 sets of 8 to 10 reps at 60 to 70% of 1RM for 11 exercises using Nautilus machines, and increasing to 3 sets per session after the first 2 weeks. 1RM was re-assessed at four week intervals to provide for overload. Post training 1RM was reassessed after the intervention period and again 6 weeks after completion of the intervention.
- cross-training group (XTG) – training 4 times per week but alternating between aerobic and resistance training – with assessments and intensities as described for the ATG and RTG
- control group (CG) – where participants were asked not to engage in any regular training and to complete an activity log to submit for review each week.

Direct laboratory analysis of blood was undertaken for TC, Tg and HDL-C. LDL-C was estimated using Friedewald's equations. Apolipoprotein levels were not assessed.

Diet was not controlled per se, but dietary intake was recorded for 7 days prior, and repeated at week 8, week 16 and at 6 weeks after completion of the intervention. All subjects were periodically reminded not to change their dietary habits.

The only significant change in blood lipids over the exercise intervention period was decrease in Tg and a 28% increase in HDL-C for the ATG. The ATG was also the only group to record a significant change in anthropometric measures and cardiovascular fitness measures – with a 13% reduction in body fat% and a 26% increase in VO_{2max} over the 16 week intervention.

Both effects of aerobic training were transient. Body fat% and VO_{2max} reverted to baseline after 6 weeks of detraining, as did blood lipid values. RTG, XTG and CG showed no significant change in any blood lipid measure as a consequence of training.

Dietary analysis revealed no significant difference in average daily energy intake between groups or with time. Macronutrient contributions to daily energy intake remained consistent throughout the study period.

The researchers argue that the higher intensity exercise undertaken by the RTG and XTG is predominantly fuelled from glycogen stores and that the higher intensity exercise may not have resulted in the adaptations that lead to improved fat utilization during exercise.

Analysis of pooled data for all subjects revealed that changes in body fat% accounted for 40% of the variation in HDL-C, and that changes in VO_{2max} accounted for 23% of the variation in HDL-C after 16 weeks.

This study maintained rigorous control of participant's activity and diet. Despite no significant change in energy intake across the exercise intervention, no group experienced significant weight loss despite substantial increases in average daily energy expenditure for ATG, XTG and even RTG.

The study clearly demonstrated a positive (but transient) effect on HDL-C with aerobic training. However, it should be noted that the bulk of effect on HDL-C was attributed to reduction in body fat%.

Ring-Dimitriou S, von Duvillard SP, Paulweber B, Stadlmann M, LeMura LM, Peak K, et al. Nine months aerobic fitness induced changes on blood lipids and lipoproteins in untrained subjects versus controls. *European Journal of Applied Physiology*. 2007;99(3):291-9.

This randomised controlled trial investigated the effect of 9 months of regular endurance exercise training on blood lipid concentrations, aerobic fitness and running performance. The training activity was running; with intensity and duration individually specified for each subject.

The authors initially recruited 52 untrained subjects ranging in age from 35 to 55 years from an area around the University of Salzburg in Austria. They were non smokers; not being treated for any chronic

disease, hypertension or dyslipidemia; and not undertaking more than 2 hours of physical activity per week. They were randomly allocated to an Exercise Group (EG) and a Control Group (CG). All 52 subjects were initially assessed on a motor driven treadmill to estimate $\text{VO}_{2\text{peak}}$; only subjects with a $\text{VO}_{2\text{peak}} \leq 40$ ml/kg/min were selected to continue with the study. This criterion eliminated 7 female subjects from the CG. A further 9 CG subjects withdrew for a range of reasons. The EG commenced with 26 subjects but lost 6 through injury and other causes.

All subjects were "asked to maintain their usual nutritional habits throughout the study". The study's subjects commenced with mean LDL-C levels of 124.1 mg/dL in the NCEP "near optimal" classification and with relatively elevated mean HDL-C levels at 63.3 mg/dL. At commencement, no significant differences existed between the EG and CG in terms of anthropometrics, aerobic fitness or blood lipid concentrations.

For participants in the EG, the initial treadmill assessment (at 1% incline) identified a running velocity for each subject that resulted in blood lactate of 2 mmol.L⁻¹ (referred to as v-LA2). This running velocity was specified as the target training intensity. Each three months, v-LA2 was re-assessed, and training duration was increased from 2.0 hours to 2.5 hours and to 3.0 hours per week for the final 3 months. In the EG, v-LA2 was equivalent to 64 to 73% of $\text{VO}_{2\text{peak}}$ or 7.8 to 9.3 METs.

The subjects reported completing 92% of all running volume at \leq v-LA2.

No significant changes in anthropometric measures (body mass, body fat% and BMI) were identified within or between the treatment groups over the 9 months of the study. Running performance and aerobic fitness improved significantly. In the EG - mean $\text{VO}_{2\text{peak}}$ increased significantly by 24%. v-LA2, v-LA4 and maximal treadmill velocity, v-Max, all significantly increased.

The only significant change in blood lipid concentrations was an 18% reduction in the apolipoprotein B (apo-B) concentration in response to 9 months of exercise training. No other favourable lipid or lipoprotein changes were detected.

The authors note that Thompson et al (1991) found chronic regular endurance exercise of >3 hrs/week enhanced HDL-C in healthy men. They attribute their failure to elicit a similar HDL-C response to the larger range in training volume in their study and to the relatively high initial HDL-C levels of the EG.

In this study, no attempt was made to assess change in dietary habits across the period of the study. Such changes may well have confounded the true effect of the exercise intervention.

This study is yet another failed attempt to identify an aerobic training regimen capable of soliciting a consistent positive effect on a range of blood lipid and lipoproteins associated with coronary heart disease risk for the population from 35 to 55 years of age.

References

Thompson P, Cullinane E, Sady S, Flynn M, Chenevert C, Herbert P. High density lipoprotein metabolism in endurance athletes and sedentary men. *Circulation*. 1991 July 1, 1991;84(1):140-52.

Thompson P, Cullinane E, Sady S, Flynn M, Chenevert C, Herbert P. High density lipoprotein metabolism in endurance athletes and sedentary men. *Circulation*. 1991 July 1, 1991;84(1):140-52.

Wood and Haskell (1979) reported a strong correlation between chronic endurance training and healthy levels of HDL-C in men. While this association has been observed in numerous studies, the mechanism is not well understood.

This randomised controlled trial compared the HDL-C metabolism of male endurance athletes with that of sedentary men. It also examined lipase activity and fat clearance rates after a period of 28 days of controlled dietary intake.

The researchers recruited 10 endurance athletes into an active group (AG) and 10 sedentary men into a control group (CG). All subjects were healthy non-smokers who used no medications regularly. The AG subjects all ran an average of more than 48 km/week for the 12 months prior to the study. The CG subjects exercised once per week or less for the preceding 12 months.

The AG ran a distance of 8 to 16 km/day for 14 days prior to and for the 28 days of the study. The CG subjects were required to maintain their traditional sedentary behaviour throughout the study period.

All subjects were placed on a controlled diet for the 28 day study period. The diet was designed to approximate the average U.S. diet, but with individualised caloric content to match estimates based on Harris-Benedict equations with activity factors to suit the treatments. The diet provided 17% of energy from protein, 43% from carbohydrate and 40% from fat (with 18% from saturated, 16% from mono-unsaturated and 6% from polyunsaturated fats).

Blood samples at days 1, 8, 15-28 were analysed for TC, HDL-C, Tg, and Apolipoproteins A-I, A-II and B. The sub fractions HDL₂ and HDL₃ of HDL-C were determined according to particle density. LDL-C was estimated using Friedewald's formula.

HDL-C kinetics were studied on days 15-28. The average of each subject's lipid values over the 14 days was used in subsequent data analysis. The endurance athletes were found to maintain average HDL-C levels significantly higher (41% higher) than the sedentary men. Tg were maintained 45% lower in the active men. There were no observed significant differences in apo-AI and apo-AII synthetic rates between endurance trained and sedentary men.

All subjects were transfused with autologous iodine labelled HDL at day 14 to enable tracking of the rate of catabolism of HDL particles over the 14 days of blood sampling. The fractional catabolic rate of HDL was significantly lower for the endurance trained men.

Intravenous fat tolerance was assessed on day 29. Each subject was transfused with 1 mL/kg body weight of a 10% fat emulsion over 1 minute. Plasma Tg were measured every 5 minutes for the next 40 minutes to obtain an estimate of each subject's triglyceride clearance rate.

Despite an absence of significant differences in lipoprotein lipase (LPL) and hepatic triglyceride lipase (HTGL) between the endurance trained and sedentary men, the clearance rate for intravenous fat was 80% faster for the endurance trained men.

The researchers suggest that enhanced HDL particle survival in endurance trained athletes results from more efficient catabolism of triglyceride rich lipoproteins (TRL) and delivery of lipid to HDL that prolongs the survival of the major HDL proteins. They note "HDL-C was inversely related to triglyceride concentrations in both subject groups and directly related to the rate of clearance of intravenous fat".

The absence of a significant difference in LPL between the groups surprised the researchers. LPL is commonly regarded as the rate-limiting enzyme in the hydrolysis of TRL, hence their expectation that endurance athletes would present with significantly higher LPL levels. They attribute this unexpected result to study design issues. They only sampled for LPL after completion of the fat-tolerance testing on day 29, and speculate that the test may have affected plasma LPL concentrations.

They also speculate that the differences in plasma volume between the trained and sedentary subjects may have affected the result. After adjusting LPL and HTGL concentrations to account for the higher plasma volume of the trained athletes, the researchers were able to "establish" significant differences between the subject groups that were consistent with the observed clearance rates for intravenous fat.

This study used a diet selected to match the average U.S. diet. It provided 18% of energy from saturated fat. The authors note that the controlled diet in this study tended to exaggerate HDL differences between the study groups. A repeat of this study following the Clinical Guidelines of NCEP ATP III, which recommend <7% of energy from saturated fat, would be an interesting follow-up study.

References

Expert Panel on Detection Evaluation Treatment of High Blood Cholesterol in Adults. Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA: The Journal of the American Medical Association. 2001 May 16, 2001;285(19):2486-97. DOI: 10.1001/jama.285.19.2486

Tjonna AE, Lee SJ, Rognmo O, Stolen TO, Bye A, Haram PM, et al. Aerobic Interval Training Versus Continuous Moderate Exercise as a Treatment for the Metabolic Syndrome: A Pilot Study. Circulation. 2008 July 22, 2008;118(4):346-54. DOI: 10.1161/circulationaha.108.772822

This paper is one of a series of papers from researchers at the Norwegian University of Science and Technology at Trondheim. The paper describes a randomised controlled trial involving 32 middle aged subjects, all diagnosed with metabolic syndrome as per the WHO definition. This trial is a challenging one in that the subjects were suffering from a range of “connected” conditions and were using a disparate range of medications including anti-hypertensives, α and β -blockers, statins and metformin.

The aim of the study was to examine the effect of aerobic exercise intensity on numerous diagnostic measures of metabolic syndrome, including indicators of dyslipidemia and factors influencing those indicators, including analysis of muscle and adipose tissue samples.

Subjects were allocated to a control (C), continuous moderate aerobic exercise (CME) or an aerobic interval training (AIT) group. The subjects in the treatment groups undertook supervised treadmill training for 16 weeks, 3 times per week. Each CME session involved 10 mins warm-up then 47 mins at 70% HR_{max} . The AIT sessions involved 10 mins warm-up then 4 blocks of 4 mins @90% of HR_{max} followed by 3 mins active recovery at 70% of HR_{max} .

Both exercise treatments resulted in significant reductions in body mass, BMI and waist circumference; and increases in relative VO_{2max} . However, neither exercise treatment had any significant effect on Tg, LDL-C or TC. HDL-C increased \approx 25% in the AIT group but was not affected significantly by CME. Blood analysis also revealed significant increases in circulating adiponectin with both CME and AIT. The authors attribute the increases in this adipocyte-secreted hormone to a reduction in visceral obesity following the exercise treatments.

From the tissues samples of skeletal muscle, the authors observed a 3-fold increase in insulin receptors with AIT, but no effect from CME. Fat tissues samples from the gluteal fat pad revealed significant decreases in fatty acid synthase with both CME and AIT. Both of these effects are likely to contribute to favourable alterations to lipid metabolism.

Papers from this team at Trondheim extend beyond simply observing effect of exercise on lipid and lipoprotein levels and tend to explore mechanisms. The authors conclude that high intensity exercise interventions show promise for those diagnosed with metabolic syndrome but caution that the injury risk of intense training protocols has not been assessed.

References

WHO. Report of a WHO Consultation: Definition of the Metabolic Syndrome, Diagnosis, and Classification of Diabetes Mellitus and Its Complications, I: Diagnosis and Classification of Diabetes Mellitus. Geneva, Switzerland: World Health Organization, Department of Non-communicable Disease Surveillance 1999.

Williams PT, Wood PD, Krauss RM, Haskell WL, Vranizan KM, Blair SN, et al. Does weight loss cause the exercise-induced increase in plasma high density lipoproteins? Atherosclerosis. 1983;47(2):173-85.

The Framingham Study (Kannel et al 1979), a longitudinal study of over 5000 men and women in the USA, identified that lean individuals have higher HDL-C levels than obese individuals. Research prior to 1979 was identifying anti-arthrogenic effects of elevated HDL-C suggesting mechanisms that might explain the observed association. By 1981, the clinical significance of the HDL subfractions had been described by Miller et al.

Despite these early appreciations of the significance of the HDL subfractions many subsequent exercise studies have focused on an association between endurance training and total HDL-C levels, without regard for the differential effects of the HDL2 and HDL3 subfractions, nor on significance of weight loss in the exercise effect.

This randomised controlled trial sought to examine the effect of prolonged endurance training on weight loss and HDL-C fractionation in middle-aged sedentary men,

The researchers recruited 81 healthy, sedentary men aged between 35 and 55 years of age who were assigned at random to an exercise group (EG, n=48) and a control group (CG, n=33). Subjects in the CG were asked to maintain their sedentary lifestyle for one year. Subjects in the EG participated in a supervised running program that met 3 to 5 days per week for one year. Training intensity was specified as 70 to 85% of HR_{max} . Subjects recorded frequency and distance of all running activity. Diet was self managed.

Over the study period, the EG lost significant body mass, mostly due to fat loss, with little change in lean body mass. Changes toward leaner body composition were actually most pronounced in those who increased total dietary caloric intake. An observation that is perhaps not so surprising given that caloric intake was positively correlated with miles run.

The researchers found that processes associated with weight change produce much of the increase in HDL-C associated with moderate aerobic exercise training. They also found that most of the positive change in total HDL-C resulted from change in the anti-arthrogenic HDL2 subfraction

However, they also observed that weight loss without exercise conditioning in the control group did “not necessarily produce change in plasma HDL-C concentrations”. This observation is not supported by any statistical analysis, and may be ill founded. In fact, the CG had an average weight gain of 1.1 kg over the study period, so presumably a minority experienced weight loss. The reasons for the weight loss in this subsample of the control group are not explored by these researchers, and may well have resulted from disease, lifestyle change or other cause, rather than caloric restriction or manipulation of fat intake, the lifestyle changes most often implemented as an initial treatment for dyslipidemia.

Wooten JS, Phillips MD, Mitchell JB, Patrizi R, Pleasant RN, Hein RM, et al. Resistance Exercise and Lipoproteins in Postmenopausal Women. Int J Sports Med. 2011 26.01.2011;32(EFirst):7-13. DOI: 10.1055/s-0030-1268008

This randomised controlled trial examined the effect of exercise on the blood lipid and lipoprotein-cholesterol levels of obese postmenopausal women. The authors examined the effect of both a 12-week resistance training program and a single session of resistance exercise.

Few studies have specifically targeted obese postmenopausal women. The authors suggest that the failure of previous studies in this population to deliver consistent effect may also be due to failure to account for exercise-induced changes in plasma volume post-exercise and for the use of hormone replacement therapy (HRT).

The researchers recruited 24 sedentary, obese (BMI 30 – 40 kg/m²), postmenopausal women aged between 60 and 70 years. They excluded women undergoing HRT.

The initial assessment of all participants involved -

- body composition
- sub-maximal treadmill stress test to 85% of age predicted maximum heart rate with ECG and blood pressure monitoring
- introduction and instruction to 10 compound resistance exercise targeting large muscle groups
- muscular strength was estimated, in terms of 8 repetition maximum (8-RM), for each exercise.

The exercise group undertook 12 weeks of resistance training – 3 days per week undertaking 3 sets of 10 exercises – the first two sets at 8-RM and the 3rd set to failure. If a participant exceeded 12 repetitions on the last set, weight for the exercise was increased on the next workout day.

The control group attended “social interaction” sessions twice per week for general health education.

All participants were requested to maintain normal diet throughout the study period.

On completion of the 12-week program, all remaining participants undertook a single resistance exercise session. Blood samples were taken immediately pre, and 24 hours post, the resistance exercise session.

A single bout of resistance exercise did not significantly modify any blood lipid concentrations 24 hours post exercise.

On completion of the 12 weeks of resistance training, there was no significant change in BMI, body mass or body composition for either the exercise or control group.

Participants in the exercise group showed significant increases in muscular strength, and the effect on blood lipids was limited to TC and LDL-C, both significantly lower in the exercise group than the control group.

The authors suggest that the observed effect on LDL-C may translate to a 3.4% reduction in the risk of a nonfatal coronary heart event in this group of women.

Interestingly, no significant acute or chronic effect of resistance exercise on HDL-C was observed. The authors contrast this with the findings of Bemben and Bemben (2000) who found a 12% increase in HDL-C after 16 weeks of resistance training with Dynabands in a study of postmenopausal women.

No exercise treatment effect on triglyceride levels confirms the findings of Bemben and Bemben (2000) and Elliot et al (2002) in other samples from the population of postmenopausal women.

The researchers selected a relatively healthy group of obese postmenopausal women – they excluded for a great many of the disorders that commonly affect obese postmenopausal women. Hence the applicability of the results is quite narrow.

The finding of no acute effect of resistance training, contrasts strongly with earlier studies of healthy trained and untrained men, where a single session of whole body, high volume resistance training has been shown to significantly reduce Tg levels and increase HDL-C. The finding of a significant effect of resistance training on LDL-C conflicts with the earlier finding of no effect in the study of Elliot et al (2002).

The confounding effect of dietary intake on the results of this study is unclear. While participants were encouraged to maintain a “normal” diet throughout the study period, their participation in the study and elevated focus on health, is likely to have lead to dietary changes that may have contributed to the observed reduction in LDL-C. The authors did no monitoring or analysis of dietary intake.

References

Bemben DA, Bemben MG. Effects of resistance exercise and body mass index on lipoprotein-lipid patterns of postmenopausal women. *The Journal of Strength & Conditioning Research*. 2000;14(1):80.

Elliott K, Sale C, Cable N. Effects of resistance training and detraining on muscle strength and blood lipid profiles in postmenopausal women. *British Journal of Sports Medicine*. 2002;36(5):340.

Abbreviations

| | |
|--------|--|
| ApoB | Apolipoprotein B |
| CHD | Coronary heart disease |
| HDL | High-density lipoprotein |
| HDL-C | High-density lipoprotein cholesterol |
| LDL | Low-density lipoprotein |
| LDL-C | Low-density lipoprotein cholesterol |
| LPL | Lipoprotein lipase |
| NCEP | National Cholesterol Education Program |
| TC | Total Cholesterol |
| VLDL | Very low-density lipoprotein |
| VLDL-C | Very low-density lipoprotein cholesterol |

ABSTRACTS

Angelopoulos T, Sivo S, Kyriazis G, Caplan J, Zoeller R, Lowndes J, et al. Do age and baseline LDL cholesterol levels determine the effect of regular exercise on plasma lipoprotein cholesterol and apolipoprotein B levels? *European Journal of Applied Physiology*. 2007;101(5):621-8. DOI: 10.1007/s00421-007-0537-1(2)

Abstract - Apolipoprotein B (apoB) concentration and age are independently associated with an increased risk for cardiovascular disease. Age is also associated with increased apoB concentration. The purpose of this study was to determine the effects of exercise on apoB and examine the association between age and lipoproteins. Forty-one sedentary individuals exercised for 6 months, four times/week for 40 min between 60 and 85% of their maximal heart rate. Lipids were determined three times: before training, 24 and 72 h after the last training session. Exercise did not alter apoB (1.2 ± 0.05 g/l vs. 1.2 ± 0.05 g/l; $P > 0.05$), or other lipids or lipoproteins. When participants were sequestered by baseline low density lipoprotein cholesterol (LDLc), total cholesterol (TC) was decreased at 24 h post (6.3 ± 0.2 mmol/l vs. 6.0 ± 0.2 mmol/l, $P < 0.05$) and LDLc after 24 and 48 h post (4.3 ± 0.1 mg/dl vs. 3.9 ± 0.1 and 4.1 ± 0.2 mg/dl, $P < 0.05$) in the high LDLc group. In the low LDLc group both TC (4.4 ± 0.2 mmol/l vs. 4.6 ± 0.2 and 4.6 ± 0.2 mmol/l, $P > 0.05$) and LDLc (2.6 ± 0.1 mmol/l vs. 2.8 ± 0.1 and 2.8 ± 0.2 mmol/l, $P < 0.05$) were elevated at 24 h and remained elevated at 72 h post compared to baseline. Age does not affect apoB or lipoproteins in response to exercise. Individuals with high baseline LDLc experienced acute reduction in TC and LDLc produced by each exercise session.

Bey L, Hamilton MT. Suppression of skeletal muscle lipoprotein lipase activity during physical inactivity: a molecular reason to maintain daily low-intensity activity. *The Journal of Physiology*. 2003;551(2):673. DOI: 10.1113/jphysiol.2003.045591

Abstract - We have examined the regulation of lipoprotein lipase (LPL) activity in skeletal muscle during physical inactivity in comparison to low-intensity contractile activity of ambulatory controls. From studies acutely preventing ambulatory activity of one or both the hindlimbs in rats, it was shown that ~90–95 % of the heparin-releasable (HR) LPL activity normally present in rat muscle with ambulatory activity is lost, and thus dependent on local contractile activity. Similarly, ~95 % of the differences in LPL activity between muscles of different fibre types was dependent on ambulatory activity. The robustness of the finding that physical inactivity significantly decreases muscle LPL activity was evident from confirmatory studies with different models of inactivity, in many rats and mice, both sexes, three muscle types and during both acute and chronic (11 days) treatment. Inactivity caused a local reduction of plasma [3H] triglyceride uptake into muscle and a decrease in high density lipoprotein cholesterol concentration. LPL mRNA was not differentially expressed between ambulatory controls and either the acutely or chronically inactive groups. Instead, the process involved a rapid loss of the HR-LPL protein mass (the portion of LPL largely associated with the vascular endothelium) by an actinomycin D-sensitive signalling mechanism (i.e. transcriptionally dependent process). Significant decreases of intracellular LPL protein content lagged behind the loss of HR-LPL protein. Treadmill walking raised LPL activity ~8-fold ($P < 0.01$) within 4 h after inactivity. The striking sensitivity of muscle LPL to inactivity and low-intensity contractile activity may provide one piece of the puzzle for why inactivity is a risk factor for metabolic diseases and why even non-vigorous activity provides marked protection against disorders involving poor lipid metabolism.

Durstine JL, Grandjean PW, Davis PG, Ferguson MA, Alderson NL, DuBose KD. Blood lipid and lipoprotein adaptations to exercise: a quantitative analysis. *Sports Medicine*. 2001;31(15):1033-62.

Abstract: Dose-response relationships between exercise training volume and blood lipid changes suggest that exercise can favourably alter blood lipids at low training volumes, although the effects may not be observable until certain exercise thresholds are met. The thresholds established from cross-sectional literature occur at training volumes of 24 to 32km (15 to 20 miles) per week of brisk walking or jogging and elicit between 1200 to 2200 kcal/wk. This range of weekly energy expenditure is associated with 2 to 3 mg/dl increases in high-density lipoprotein-cholesterol (HDL-C) and triglyceride (TG) reductions of 8 to 20 mg/dl. Evidence from cross-sectional studies indicates that greater changes in HDL-C levels can be expected with additional increases in exercise training volume. HDL-C and TG changes are often observed

after training regimens requiring energy expenditures similar to those characterised from cross-sectional data. Training programmes that elicit 1200 to 2200 kcal/wk in exercise are often effective at elevating HDL-C levels from 2 to 8 mg/dl, and lowering TG levels by 5 to 38 mg/dl. Exercise training seldom alters total cholesterol (TC) and low-density lipoprotein-cholesterol (LDLC). However, this range of weekly exercise energy expenditure is also associated with TC and LDL-C reductions when they are reported. The frequency and extent to which most of these lipid changes are reported are similar in both genders, with the exception of TG. Thus, for most individuals, the positive effects of regular exercise are exerted on blood lipids at low training volumes and accrue so that noticeable differences frequently occur with weekly energy expenditures of 1200 to 2200 kcal/wk. It appears that weekly exercise caloric expenditures that meet or exceed the higher end of this range are more likely to produce the desired lipid changes. This amount of physical activity, performed at moderate intensities, is reasonable and attainable for most individuals and is within the American College of Sports Medicine's currently recommended range for healthy adults.

Elliott K, Sale C, Cable N. Effects of resistance training and detraining on muscle strength and blood lipid profiles in postmenopausal women. *British Journal of Sports Medicine*. 2002;36(5):340.

Abstract:

Objectives: To study the effects of eight weeks of supervised, low intensity resistance training (80% of 10 repetition maximum (10RM)) and eight weeks of detraining on muscle strength and blood lipid profiles in healthy, sedentary postmenopausal women.

Subjects: Fifteen postmenopausal women, aged 49–62 years, took part in the study. Subjects were assigned to either a control ($n = 7$) or training ($n = 8$) group. The training regimen consisted of three sets of eight repetitions of leg press, bench press, knee extension, knee flexion, and lat pull-down, three days a week at 80% of 10RM. Dynamic leg strength, 10RM, and blood lipid profiles (total cholesterol (TC), low and high density lipoprotein cholesterol (LDL-C, HDL-C), triglycerides, and very low density lipoprotein cholesterol (VLDL-C)) were measured at baseline, after eight weeks of training, and after a further eight weeks of detraining.

Results: Eight weeks of resistance training produced significant increases in knee extension ($F_{1,13} = 12.60$; $p < 0.01$), bench press ($F_{1,13} = 13.79$; $p < 0.01$), leg press ($F_{1,13} = 15.65$; $p < 0.01$), and lat pull-down ($F_{1,13} = 16.60$; $p < 0.005$) 10RM strength tests. Although 10RM strength decreased after eight weeks of detraining, the results remained significantly elevated from baseline measures. Eight weeks of training did not result in any significant alterations in blood lipid profiles, body composition, or dynamic isokinetic leg strength. There were no significant differences in any of the variables investigated over the 16 week period in the control group.

Conclusions: These data suggest that a short, low intensity resistance training programme produces substantial improvements in muscle strength. Training of this intensity and duration was not sufficient to produce significant alterations in blood lipid concentrations.

LeMura LM, von Duvillard SP, Andreacci J, Klebez JM, Chelland SA, Russo J. Lipid and lipoprotein profiles, cardiovascular fitness, body composition, and diet during and after resistance, aerobic and combination training in young women. *European Journal of Applied Physiology*. 2000;82(5):451-8. DOI: 10.1007/s004210000234

Abstract: The purpose of this study was to evaluate the effects of various modes of training on the time-course of changes in lipoprotein-lipid profiles in the blood, cardiovascular fitness, and body composition after 16 weeks of training and 6 weeks of detraining in young women. A group of 48 sedentary but healthy women [mean age 20.4 (SD 1) years] were matched and randomly placed into a control group (CG, $n=12$), an aerobic training group (ATG, $n=12$), a resistance training group (RTG, $n=12$), or a cross-training group that combined both aerobic and resistance training (XTG, $n=12$). The ATG, RTG and XTG trained for 16 weeks and were monitored for changes in blood concentrations of lipoprotein-lipids, cardiovascular fitness, body composition, and dietary composition throughout a 16 week period of training and 6 weeks of detraining. The ATG significantly reduced blood concentrations of triglycerides (TRI) ($P < 0.05$) and significantly increased blood concentrations of high density lipoprotein-cholesterol (HDL-C) after 16 weeks of training. The correlation between percentage fat and HDL-C was 0.63 ($P < 0.05$), which explained 40% of the variation in HDL-C, while the correlation between maximal oxygen uptake ($\dot{V}O_{2max}$) and HDL-C was 0.48 ($P < 0.05$), which explained 23% of the variation in HDL-C. The ATG increased $\dot{V}O_{2max}$ by 25% ($P < 0.001$) and decreased percentage body fat by 13% ($P < 0.05$) after 16 weeks. Each of the alterations in the ATG had disappeared after the 6 week detraining period. The

concentration of total cholesterol (TC), TRI, HDL-C and low density lipoprotein-cholesterol in the blood did not change during the study in RTG, XTG and CG. The RTG increased upper and lower body strength by 29% ($P < 0.001$) and 38%, respectively. The 6 week detraining strength values obtained in RTG were significantly greater than those obtained at baseline. The XTG increased upper and lower body strength by 19% ($P < 0.01$) and 25% ($P < 0.001$), respectively. The 6 week detraining strength values obtained in XTG were significantly greater than those obtained at baseline. The RTG, XTG and CG did not demonstrate any significant changes in either $\dot{V}O_{2\max}$ or body composition during the training and detraining periods. The results of this study suggest that aerobic-type exercise improves lipoprotein-lipid profiles, cardiorespiratory fitness and body composition in healthy, young women, while resistance training significantly improved upper and lower body strength only.

Ring-Dimitriou S, von Duvillard SP, Paulweber B, Stadlmann M, LeMura LM, Peak K, et al. Nine months aerobic fitness induced changes on blood lipids and lipoproteins in untrained subjects versus controls. *European Journal of Applied Physiology*. 2007;99(3):291-9.

Abstract - Regular endurance exercise has favourable effects on cardiovascular risk factors. However, the impact of an exercise-induced change in aerobic fitness on blood lipids is often inconsistent. The purpose of this study was to investigate the effect of nine consecutive months of training on aerobic fitness and blood lipids in untrained adults. Thirty subjects 35–55 years of age (wt: 73.1 ± 13.6 kg, height 171.1 ± 9.0 cm, %bodyfat: $24.6 \pm 6.3\%$, 14 males and 16 females) were randomly assigned to an exercise (EG) ($N = 20$) and control (CG) ($N = 10$) group. All subjects completed an incremental treadmill test, anthropometric measurements, and venous blood sample collection before and after the 9 months of exercise. Participants in the exercise group were supervised and adjusted for improvements in running performance, whereas no change was administered for the control group. One-way and multivariate ANOVA was conducted to determine significant differences in means for time and group in selected variables [body mass, % body fat, BMI; $VO_{2\text{peak}}$, km/h at 2.0 (v-LA2) and 4.0 (v-LA4) mmol.L⁻¹ blood lactate (LA) concentration, km/h of the last load (v-max); TC, LDL-C, HDL-C, TG, Apo B, Apo A-I, and Lp (a)]. Correlation coefficients and multivariate regression analysis was used to determine the association between aerobic fitness and blood lipids. The exercise group improved significantly ($P < 0.0001$) in $VO_{2\text{peak}}$, v-LA2, v-LA4, v-max and exhibited a significant decrease in Apo B ($P < 0.04$) compared to the control group (NS). In 9 months, E achieved 24% increase in $VO_{2\text{peak}}$ and 18% reduction in Apo B, denoting the impact of cardiovascular fitness on cardiovascular risk.

Thompson P, Cullinane E, Sady S, Flynn M, Chenevert C, Herbert P. High density lipoprotein metabolism in endurance athletes and sedentary men. *Circulation*. 1991 July 1, 1991;84(1):140-52.

Abstract:

BACKGROUND. Endurance athletes have higher high density lipoprotein (HDL) concentrations than sedentary controls. To examine the mechanism for this effect, we compared HDL apoprotein metabolism in 10 endurance athletes aged 34 ± 6 years (mean \pm SD) and 10 sedentary men aged 36 ± 8 years. **METHODS AND RESULTS.** Subjects were maintained on controlled diets for 4 weeks, and metabolic studies using autologously labeled ¹²⁵I HDL were performed during the final 2 weeks. Lipids and lipoproteins were measured daily during these 2 weeks, and the average of 14 values was used in the analysis. HDL cholesterol (58 ± 14 versus 41 ± 10 mg/dl), HDL2 cholesterol (26 ± 10 versus 12 ± 8 mg/dl), and apolipoprotein A-I (apoA-I) (144 ± 18 versus 115 ± 22 mg/dl) were higher in the athletes, whereas triglyceride concentrations (60 ± 18 versus 110 ± 48 mg/dl) were lower (p less than 0.01 for all). Postheparin lipoprotein lipase activity was not different, but hepatic triglyceride lipase activity was 27% lower (p less than 0.06) in the athletes. The athletes' mean clearance rate of triglycerides after an infusion of Travamulsion (1 ml/kg) was nearly twofold that of the inactive men (5.8 ± 1.5 versus $3.2 \pm 0.9\%$ /min, p less than 0.001). There was no differences in HDL apoprotein synthetic rates, whereas the catabolic rates of both apo A-I (0.15 ± 0.02 versus 0.22 ± 0.05 pools per day, p less than 0.01) and apolipoprotein A-II (apo A-II) (0.15 ± 0.02 versus 0.20 ± 0.04 pools per day, p less than 0.05) were reduced in the trained men. Apo A-I and apo A-II half-lives correlated with HDL cholesterol in each group (r greater than 0.76, p less than 0.05 for all) but not consistently with lipase activities or fat clearance rates. This relation between apoprotein catabolism and HDL cholesterol was strongest at HDL cholesterol concentrations of less than 60 mg/dl. **CONCLUSIONS.** We conclude that higher HDL levels in active men are associated with increased HDL protein survival. The mechanisms mediating this effect require better definition, and other factors appear to contribute to HDL cholesterol and protein concentrations among individual subjects.

Tjonna AE, Lee SJ, Rognmo O, Stolen TO, Bye A, Haram PM, et al. Aerobic Interval Training Versus Continuous Moderate Exercise as a Treatment for the Metabolic Syndrome: A Pilot Study. *Circulation*. 2008 July 22, 2008;118(4):346-54. DOI: 10.1161/circulationaha.108.772822

Abstract:

Background— Individuals with the metabolic syndrome are 3 times more likely to die of heart disease than healthy counterparts. Exercise training reduces several of the symptoms of the syndrome, but the exercise intensity that yields the maximal beneficial adaptations is in dispute. We compared moderate and high exercise intensity with regard to variables associated with cardiovascular function and prognosis in patients with the metabolic syndrome.

Methods and Results— Thirty-two metabolic syndrome patients (age, 52.3±3.7 years; maximal oxygen uptake [VO₂max], 34 mL.kg⁻¹.min⁻¹) were randomized to equal volumes of either moderate continuous moderate exercise (CME; 70% of highest measured heart rate [Hfmax]) or aerobic interval training (AIT; 90% of Hfmax) 3 times a week for 16 weeks or to a control group. VO₂max increased more after AIT than CME (35% versus 16%; P<0.01) and was associated with the removal of more risk factors that constitute the metabolic syndrome (number of factors: AIT, 5.9 before versus 4.0 after; P<0.01; CME, 5.7 before versus 5.0 after; group difference, P<0.05). AIT was superior to CME in enhancing endothelial function (9% versus 5%; P<0.001), insulin signaling in fat and skeletal muscle, skeletal muscle biogenesis, and excitation-contraction coupling and in reducing blood glucose and lipogenesis in adipose tissue. The 2 exercise programs were equally effective at lowering mean arterial blood pressure and reducing body weight (−2.3 and −3.6 kg in AIT and CME, respectively) and fat.

Conclusions— Exercise intensity was an important factor for improving aerobic capacity and reversing the risk factors of the metabolic syndrome. These findings may have important implications for exercise training in rehabilitation programs and future studies.

Williams PT, Wood PD, Krauss RM, Haskell WL, Vranizan KM, Blair SN, et al. Does weight loss cause the exercise-induced increase in plasma high density lipoproteins? *Atherosclerosis*. 1983;47(2):173-85.

Abstract: Studies showing an increase in plasma concentration of high density lipoprotein cholesterol (HDL-C) with moderate exercise have usually rejected the role of body weight change in the HDL-C raising process, ostensibly because the amount of weight lost has been negligible. To investigate HDL-C changes more thoroughly, we followed initially sedentary middle-aged men randomly assigned to either a moderate running (n = 36) or a sedentary control (n = 28) group for one year. Among runners, one-year changes in plasma HDL-C concentrations correlated strongly with their body weight changes ($r = -0.53$, $P < 0.001$). Curve-fitting procedures and regression analysis suggested that processes associated with weight change produce much of the plasma HDL-C changes induced by moderate exercise and that changes in HDL-C concentration predominantly reflect changes in the reputedly anti-atherogenic HDL₂ sub-component. Further, the interaction between weight change and plasma HDL-C concentration was significantly different ($P < 0.001$) in exercisers and controls suggesting that the metabolic consequences of exercise-induced weight change are different from the consequences of weight change in the sedentary state.

Wooten JS, Phillips MD, Mitchell JB, Patrizi R, Pleasant RN, Hein RM, et al. Resistance Exercise and Lipoproteins in Postmenopausal Women. *Int J Sports Med*. 2011 26.01.2011;32(EFirst):7-13. DOI: 10.1055/s-0030-1268008

Abstract - The specific aims of this study were to quantify the effects of 12 weeks of resistance training, as well as a single session of resistance exercise on lipids and lipoproteins in obese, postmenopausal women. 21 obese, postmenopausal women, not on hormone replacement therapy (age = 65.9 ± 0.5 yr; BMI = 32.7 ± 0.8 kg/m²), were randomly assigned to control (n = 12) and exercise (n = 9) groups matched for age and BMI. For 12 weeks, 3 days/week, the exercise group performed 10 whole body resistance exercises (3 sets at 8-RM). Fasting (10 h) blood samples were collected immediately prior to and 24 h after the first and last exercise and control session. Serum was assayed for concentrations of total cholesterol, triglycerides, LDL-C, HDL-C, HDL₂-C, HDL₃-C, non-HDL-C and TC:HDL and LDL:HDL ratios. The exercise group exhibited a significant ($P < 0.01$) improvement in muscular strength, but no change in BMI, body mass or body composition post-training. Total cholesterol, LDL-C and non-HDL-C were significantly ($P < 0.05$) lower in the exercise compared to the control group following the 12 weeks of resistance training. Whole body resistance training provides obese, postmenopausal women a non-pharmacological approach for the reduction of lipid and lipoprotein-cholesterol concentrations.